To Sir Wm. T. Gairdner, K.C.B.
the Author's kindest regards

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An Address
ON
MENINGITIS.

Delivered before the Birkenhead Medical Society.

BY
JAMES BARR, M.D.,
Physician, Liverpool Royal Infirmary; Lecturer on Clinical Medicine,
University College, Liverpool.

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AN ADDRESS

ON

MENINGITIS.

Gentlemen,—When I selected the subject of Meningitis for this address I was well aware that I was coming before an audience of practical men who would appreciate any serious attempt to reduce the fearful mortality which has hitherto attended this disease. I may now speak of it as a protean malady, because there are not only many varieties of the disease, but many of the symptoms—more or less common to all the varieties—are very varied in their obtrusiveness, and very closely resemble those of other acute diseases, so that this affection is frequently not recognised at an early stage.

In the *Liverpool Medico-Chirurgical Journal* for January, 1891, I published an account of 13 cases, with only one death. This exceptionally good result was no doubt largely due to fortuitous circumstances, to the fact that the cases came early under my observation, and to the fact that I then excluded all tuberculous cases. However under any circumstances 12 recoveries from meningitis was a result with which anyone might feel pleased. Since then I have copious notes of 26 cases with 10 deaths; of these

3 died within 24 hours of admission.
1 ,, ,, 48 ,, ,,
2 ,, ,, 72 ,, ,,
1 ,, ,, 96 ,, ,,

Moreover three of the deaths were from tuberculous meningitis, and one from cerebro-spinal fever.

Of all the cases 20 were males and 6 females. The ages of the males ranged from 5 to 30, the average age being 16.6; of the females from 2 to 28, the average age being 15 years. I have seen many other cases in the practice of my colleagues and in consultation, but these 26 cases will suffice "to point a moral or adorn a tale."

I have taken the subject of meningitis in its widest sense to include all inflammatory processes of the meninges, however originated; but pachymeningitis, externa and interna, chiefly come under the care of the surgeon and alienist respectively, so that I need not do more than mention them here except to point out that in many cases of extension of inflammation from the middle ear to the pia the dura mater is also involved, and over the petrous bone all the membranes may be matted together. Here again the treatment is largely surgical.

We shall now come to the subject proper of the paper, leptomeningitis. The classification of the various types of disease which are grouped under this generic head is still very indefinite, and an anatomical basis is frequently adopted accordingly as the base, the vertex, or the ventricles of the brain are chiefly affected. This division is sometimes convenient, but not of much practical importance, except for explaining variations of clinical symptoms, as in very few cases is the affection limited to one particular area. Of much more value would be a classification founded on the etiology, and in this direction we may hope in the future to learn much from bacteriology. So far it has not taught much more, except in the case of cerebro-spinal fever, than that a great variety of micro-organisms have been found; consequently we have still to speak, as our fathers did before us, of simple, purulent, tuberculous, and syphilitic meningitis. All these varieties present many features in common, so our first object should be to determine whether the patient be suffering from meningitis, and then as far as possible settle its location, and from a careful study of the history and symptoms of the case determine its nature.

Etiology.—The disease, especially the tuberculous variety, is most common in childhood, but the incidence in adult life is greater than is generally supposed. In the *Liverpool Medico-Chirurgical Journal* for July, 1885, the late Dr. Oxley recorded 63 cases with 62 deaths, all under the age of 10 years. These occurred in a practice of 22 years' duration, which was almost exclusively amongst children. It is more prevalent among males than females. Among other predisposing causes may be mentioned heredity, bad sanitary surroundings, deficient nourishment, and mental over-work. However predisposed the individual, the lighting up of the inflammation must in every case be ascribed to an infective process. In fact it is doubtful whether such causes as insolation, traumatism, with even laceration and hæmorrhage can be looked upon as other than remote agents. The meninges, like other serous membranes, injured or uninjured, are liable to inflammatory processes which may be started by various pathogenic micro-organisms or their toxins. It has been often stated that the presence of tubercles in the meninges is capable of setting up irritation and inflammation, but it is more highly probable that the tubercles and meningitis are the results of a common cause, the tubercle bacili and their toxin. In fact in many cases the inflammation is out of all proportion to the number of tubercles, and the clinical symptoms are often more pronounced than would be readily accounted for by *post-mortem* appearances, so that in such cases there must be a general toxæmia, the brunt of which is borne by the nervous system. Every physician of experience when viewing a necropsy in many a case of tuberculous meningitis, must have asked himself, Why did this child die? a question which would be often difficult to answer if he had no knowledge of the previous nerve storm. The explosion has been kindled by a toxin which the grosser pathology does not reveal, but which should not be beyond our powers to render harmless. We must not cry *non possumus*, that tuberculous meningitis is necessarily a fatal disease; we must not apply such balm to our consciences as that the disease is incurable; "that blessed word Mesopotamia" must give way to healthy action. In future we will not have to ask the question in such cases why did the patient die, but why did we allow him to die? Many of the other micro-organisms, such as the streptococci, which attack the meninges are much more deadly in their effects. A meningitis which has reached a suppurative stage must be looked upon as almost necessarily fatal unless this result can be averted by surgical interference. Micro-organisms may reach the meninges through different paths, such as middle ear disease, necrosis of the petrous bone, disease of the sphenoid and ethmoid bones and upper nasal cavities. A tumour or abscess of the brain, or a septic thrombus may start a local or general inflammation. They may also be conveyed by the blood, or travel along the lymph channels. It is occasionally caused by many of the acute infectious diseases such as pneumonia, erysipelas, measles, scarlet fever, typhoid fever, septicæmia, ulcerative endocarditis, etc. It must often be doubtful as to whether the micro-organisms or their toxins are the exciting causes. In tetanus, for example, it is highly probable that the active congestion produced by the toxin would soon be worthy of the name of inflammation if the patient survived. The rheumatic poison, which is not generally ascribed to a micro-organism, may give rise to a basilar inflammation just as it lights up a pericarditis. I believe the pneumococcus is a not infrequent source of infection, but I cannot agree with those who assert that such cases are usually suppurative. This is a short-lived organism which is very susceptible to treatment. To an allied organism, the diplococcus intracellularis of Weichselbaum, cerebro-spinal fever is due. In a recent epidemic of this disease in Boston, Professor Osler found this organism in the cerebro-spinal fluid obtained by lumbar puncture. Councilman and others have also shown that the diplococcus intracellularis is the cause of this disease. To the influenza bacillus meningitis has been not infrequently ascribed, and we know that, independently of this affection, it has often caused optic neuritis and middle ear disease.

Syphilis as a rule gives rise to a local rather than a general inflammation.

Meningitis associated with external wounds is usually septic, and, notwithstanding the advances of surgery, is often fatal. In such cases the presence of micro-organisms is easily explained, but in most other instances they must find an entrance through the blood or lymph channels. It is highly probable that there are often organisms present in the blood and tissues which in normal conditions are destroyed, but which in states of depraved health find a suitable nidus in the vascular pia.

Leptomeningitis infantum, to which a very lengthy article has been devoted in *Ziemssen's Cyclopædia*, is probably a title which will disappear with advancing knowledge. It is chiefly used to cover those cases in early childhood which are not distinctly tuberculous, and especially such as recover. It is a simple inflammation probably of toxic origin, and may occur under very varied circumstances. We all know how many specific diseases in childhood are ushered in by convulsions and other cerebral disturbances. The delicate nervous system may be so damaged by these explosions that meningitis may be readily set up.

Pathology.—The inflammation is marked in the early stages by redness of the pia mater and slight lymph effusion into the perivascular and subarachnoid spaces. The chief incidence of the process may fall on the convexity, the base, or the ventricles. Tuberculous meningitis usually commences at the base and extends up the Sylvian fissures, and is attended by inflammation of the choroid plexus with effusion into the ventricles. When this latter is great the surface of the convolutions may be flattened and have a dry appearance. A few small opaque white miliary tubercles are found along the course of the vessels, and tubercle bacilli have been found in the subarachnoid and spinal fluid. Very often the macroscopic appearances are comparatively slight, and seem quite insufficient to account for death. When the disease is more protracted there is often a considerable amount of opaque gelatinous lymph between the optic chiasma and the pons, at the commencement of the Sylvian fissures and over the rest of the base, along the sides of the pons, on the under surface of the cerebellum and around the medulla. The lymph over the fourth ventricle may be so dense that the foramen of Majendie gets closed, and there is no escape for the ventricular fluid, which then accumulates and distends the ventricles, giving rise to the condition termed acute hydrocephalus. There is a considerable amount of subarachnoid fluid in the lacunæ of the base, and the sheaths of the optic nerves may be much distended. My own limited observation agrees with that of Gowers that "the inflammation is never actually purulent," but Huguénin in *Ziemssen's Cyclopædia* asserts that in protracted cases "general purulent inflammation of the pia is the second great lesion of this disease."

In a large proportion of cases of tuberculous meningitis in children the disease is primary, or at least in a considerable number of *post-mortem* examinations no other primary source of infection has been found. Possibly if the bowel were more systematically examined in these cases a source would be then occasionally discovered. I recollect being once asked to see a case of supposed typhoid fever, which I felt confident was one of meningitis. I was afterwards told that at the *post-mortem* examination two typhoid ulcers were found in the ileum, but I have never felt absolutely convinced that those ulcers were not tuberculous. In adults this disorder is invariably secondary to tuberculous deposits in other organs.

We occasionally get a simple basilar meningitis which runs a very protracted course, six to eight weeks. There is considerable effusion of lymph at the base; in two cases I have seen it almost half an inch thick in places. There is usually a large collection of fluid in the ventricles owing to obstruction to the exit, and to inflammation of the choroid plexus. There is considerable fever, and the disease starts with a very acute onset, and as the mental symptoms are not pronounced it is occasionally mistaken for typhoid fever. The pathology is still unsettled. In some cases the disease runs a more subacute and even chronic course, "with formation of indurated patches of connective tissue, strangulation and lesion of the nerves of the base, but without hydrocephalus."[1] I believe I have a case of this kind under my care in the Royal Infirmary at present. It is that of a bright, intelligent, healthy-looking, well-nourished servant girl, aged 21. She was at service in Manchester, and nine weeks before admission to the infirmary she began to suffer from violent headache especially about the temples and forehead, and vomiting after every article of food which she took. There was slight mental confusion, such as forgetting whether she had seasoned the food which she was cooking, but these failures of memory were readily corrected by taste. She continued at her work and had no medical advice for four weeks, when getting very weak she returned to her home in Cumberland. The vomiting and headache continued for two weeks more, when it ceased, and she then became suddenly blind. At the end of three more weeks she was sent by her medical attendant to the Liverpool Eye and Ear Infirmary on account of loss of sight. Mr. Shears found advanced optic atrophy in both eyes and almost absolute blindness. He sent her on to me. On admission there were also paresis of the left abducens oculi nerve, weakness in the grasp of the right hand, and absence of the knee-jerks. My colleagues and self at first diagnosed the case as one of cerebellar tumour, but I now look upon it as a case of chronic basal meningitis involving the optic nerves and extending up the left Sylvian fissure. The grasp of the right hand is now as strong as that of the left, and the paresis of the left external rectus muscle has disappeared, but the knee-jerks are still absent. In short she is mentally and physically quite well except for complete blindness.

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[1] Huguénin, in *Ziemssen's Cyclopædia.*

About twelve years ago I had under my care a somewhat similar but more severe and more protracted case in a young man, in whom the eye symptoms were the first to attract attention. He consulted Mr. Bickerton to see if he could give him glasses to correct his failing sight as he was soon about to be married. Mr. Bickerton found advanced double optic neuritis, and sent him to me. The meningits extended down the cord as far as the cervical enlargement, and involved the brachial plexus on the left side. The illness lasted about six months, but with the exception of total blindness he got well, and I believe he is still alive.

Basal inflammation may arise from diseased bone, abscesses, and growths; and that associated with influenza usually starts here, but is not confined to this region.

Meningitis arising in connection with insolation, pneumonia, erysipelas, septicæmia, ulcerative endocarditis, or any of the acute specific diseases usually begins in the convexity. In cerebro-spinal fever the convexity, base, ventricles, and spinal meninges are all affected, and in severe cases the disease rapidly progresses to the formation of pus which is situated in the subarachnoid spaces and does not float freely in the cerebro-spinal fluid. This has been an almost unknown, or I should rather say an unrecognised disease in this neighbourhood for many years. Those who are interested in the subject will find an excellent digest by Dr. Ormerod in Clifford Allbutt's *System of Medicine.* From a paper by Captain Buchanan, of the Indian Medical Service, published in the *Dublin Journal of Medical Science*, February, 1899, this disease would seem to have a wider geographical distribution than that ascribed to it by Hirsch. Last year I made a *post-mortem* examination for the coroner of this city in what I believe was a case of this disease. The whole surface of the brain under the arachnoid was covered with a thick layer of greenish yellow creamy pus which extended down the spinal canal; there was also a considerable amount of curdy pus in the ventricles. The history in this case was that the woman had been going about her household work and getting often drunk until three days before her death.

When the meningitis arises from middle ear disease, or necrosis of bone, the dura is also affected, is usually glued to the pia, and there may be a collection of pus between it and the bone. Pus is not often free in the arachnoid cavity except in cases of compound fracture of the skull or those in which an abscess in the brain has burst.

There is often slight softening of the cortical surface, especially in purulent meningitis; and when the choroid plexus and velum interpositum are involved the ventricular walls are softened.

Symptoms.—The symptoms vary considerably according to the site and nature of the inflammation, but there are many points common to all cases, so it will suffice for our purpose to give a general description, and at the same time note the variations which occur. A series of phases in the progress of the disease have been described under the terms prodromal, irritative, pressure, and paralytic stages. This arrangement is fairly convenient for descriptive purposes if it be clearly understood that the borderland between the different stages is generally ill-defined.

The prodromal stage is chiefly marked in the tuberculous variety, and therefore as the groundwork of our picture we will take a case of primary tuberculous meningitis in a child. For two or three weeks there are vague indefinite indications of general failure in physical and mental energy, with perhaps slight loss of flesh. There is lassitude and general *malaise*, slight frontal headache aggravated by fatigue either mental or physical. The child may not complain of the headache because he desires to be left alone, apart from all noise and play. There is frequently slight giddiness, and occasionally vomiting after food, which may or may not have been of an indigestible nature. The appetite is poor. There is mental irritability, restlessness at night, grinding of teeth which may have been ascribed to worms, sleeps with eyelids not firmly closed, and often wakes up with nightmare or a scream. It is said that the disease is sometimes ushered in without any prodromal stage in the midst of perfect health, but this I do not believe. It is true the child may not have complained, but the symptoms were there plainly depicted to any observant eye. The parents excuse their faulty observation by denying the existence of signs which they failed to perceive. You may get them to admit that they thought the child rather lazy and inattentive. In this stage there is often a slight rise of the evening temperature to 99° or 99.6°. If there be primary tuberculosis in other organs the prodromal stage may be masked and shortened.

In the other forms of meningitis there is scarcely any prodromal stage, certainly not more than two or three days.

In the second or irritative stage, with which the commencement of the disease is usually dated, the symptoms are much more pronounced, the headache is severe and marked by exacerbations, there is general hyperalgesia so that the child cannot bear to be handled, there is intolerance of light and sound so that the patient lies huddled up in bed, and if left alone will probably make no complaint. His eyelids may be kept firmly closed but relax during sleep; pupils are rather contracted. There is somnolence rather than sound sleep, and muttering delirium at night. The patient may or may not complain of giddiness, but the vertigo will be generally noticed when he gets out of bed to urinate or for some other purpose by his using one hand to steady himself. There is frequent vomiting of an explosive character after food; the bowels are usually constipated and abdomen retracted. The temperature generally ranges from 99° to 102°, but may be much higher, and is very variable in its fluctuations. Pulse slow and moderately firm, as a rule under 100, but it is also liable to great fluctuations in frequency. The tongue is at first clean and moist, but afterwards becomes slightly furred. If there be any dry secretion in the nose or cracked lips the patient is frequently picking at them. The skin is dry, and *tache cérébrale* well marked.

There is now marked rigidity of the neck and retraction of the head, so that any attempt to straighten the neck or bring the chin down on the sternum causes great pain. In cases of cerebro-spinal fever there may be marked opisthotonos. There may be aphasia and muscular twitchings or local convulsions, but these symptoms are usually deferred to a later period. This stage generally lasts about a week, but under judicious treatment it may be protracted, the further downward progress delayed, and I hope in many cases averted.

In other forms of meningitis the disease is ushered in much more abruptly and with greater violence than in the tuberculous variety. There is headache, restlessness, delirium, vomiting, hyperalgesia, vertigo, early rigidity, and weakness of the limbs. The deep and superficial reflexes are as a rule increased and remain so until the pressure stage, but here again there is a liability to great variation. I have often seen the knee-jerks present one day and absent the next, and the same may be said of ankle clonus. Owing to the hyperalgesia and over-action of the flexors it is often difficult to elicit the deep reflexes. Tongue clean and moist, bowels constipated, and abdomen retracted. The temperature has a higher range than in the tuberculous form, generally from 101° to 103°, but may reach 104°, 105°, or 106°. Its chief characteristic is its great variability and sudden fluctuations. The pulse is slow, firm, and infrequent especially in the adult, it may be only 40 and is usually below 100 per minute. In purulent meningitis there is a high range of temperature without much fluctuation. In the septicæmic variety there may be very little headache, and the case passes rapidly into coma.

There is somnolence, confusion of ideas, and delirium, but when he is roused up he answers questions intelligently, and is perfectly conscious. Sometimes when an adult is roused out of active delirium and asked what is the matter, he looks confused, and answers "Oh! nothing, I have got a headache; why did you wake me"? Unlike typhoid fever the headache does not abate when the delirium sets in. There are often periods when the patient is perfectly conscious and appears much better.

In basilar meningitis there is marked rigidity of the neck and retraction of the head, but general hyperalgesia, sensitiveness of the special senses, increased reflexes, weakness, and rigidity of the limbs are more pronounced when the convexity is affected. In this latter case there may be general convulsions especially in children, the pupils are contracted, and at a later period become dilated and unequal. Optic neuritis and strabismus as well as paralysis of the other cranial nerves occur in basal mischief, but convulsions, rigidity of the muscles of mastication which I have once seen, aphasia, paralysis of one or more limbs arise from irritation or pressure on the cortex.

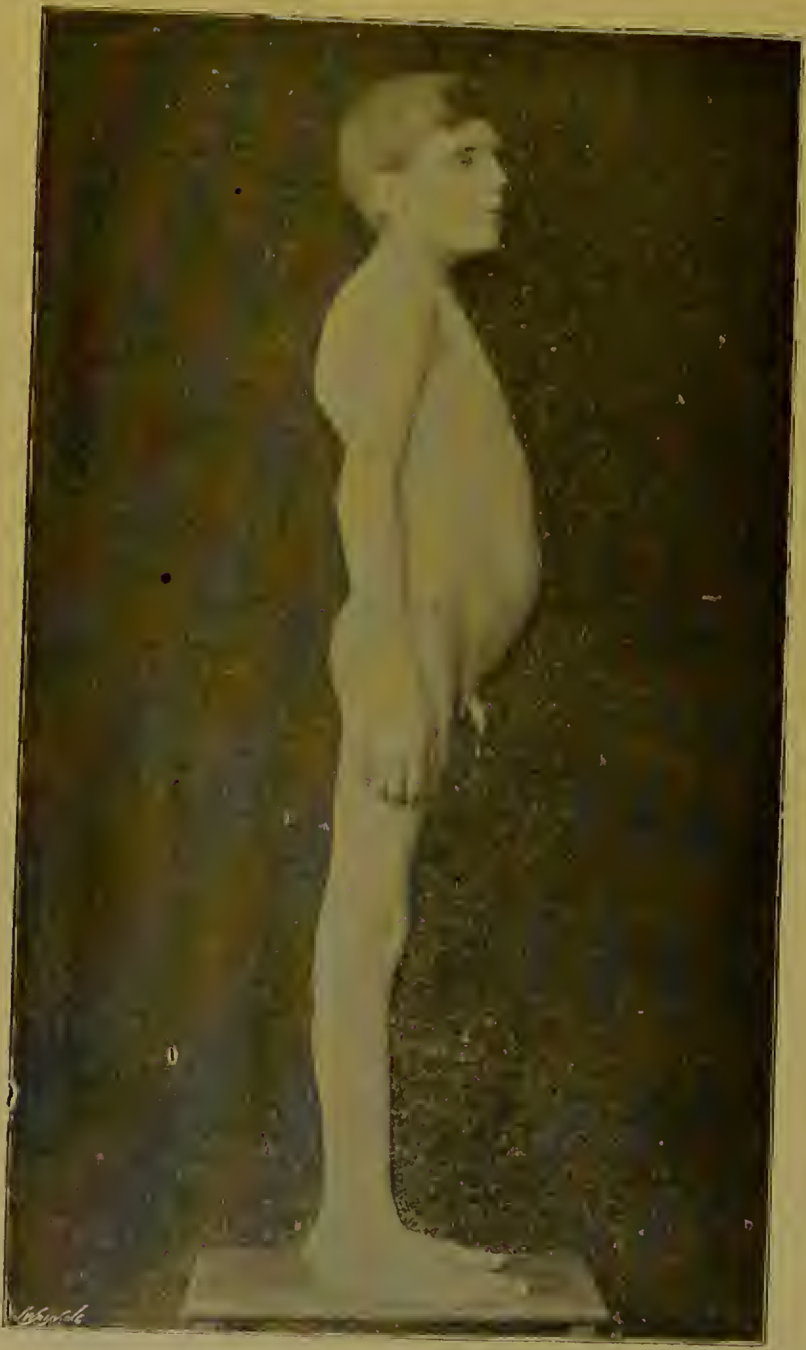
In tuberculous meningitis about the end of the second week when the case is passing into the pressure stage and the mischief extending up the Sylvian fissures there is slight optic neuritis, occasionally aphasia, muscular twitchings or slight convulsions, delirium, hyperalgesia, muscular rigidity, still intolerance of light and sound, somnolence passing into coma. Pulse small and frequent, respiration irregular, perhaps occasional vomiting, abdomen boat-shaped, bowels constipated and great emaciation; temperature very variable. About the third week the child lies very quiet and still, eyes half open with the lower half of the cornea exposed and covered with mucus or muco-pus. The child may appear asleep, but is only in a state of semi-consciousness, and there is still marked hyperalgesia, so that if you lay hold of a limb or cause any movement, the child often gives a piercing shriek —the so-called hydrocephalic cry. There are frequently slight convulsions, but with the gradually increasing fluid in the ventricles the whole body becomes paralysed and lies extended on the back. The respirations assume the Cheyne-Stokes character, the pulse becomes quite small and often irregular in rhythm. The temperature is moderate and often high before death; in some cases it is subnormal and the head moist with perspiration. From the onset of the irritative stage to death is generally from one to three weeks.

I append two illustrations of an excellent case of primary tuberculous meningitis with recovery. Those who think that all tuberculous cases die may remove the word tuberculous without in any way detracting from the value of the case. I have no absolute proof that it was tuberculous, I did not perform lumbar puncture, and did not find the tubercle bacillus. Some of my other cases of recovery were as bad, and the case of cerebro-spinal fever with recovery was worse than this patient.

In the non-tuberculous cases the symptoms during the pressure and paralytic stages vary considerably according to the site and nature of the mischief. In purulent meningitis the temperature is high and sustained, pulse rather slow and infrequent, respirations sighing, delirium perhaps slight and rapidly passing into coma. It is often difficult to distinguish such cases from cerebellar abscess, especially as both usually arise from the same cause—middle ear disease. However, the differential diagnosis is not of much practical importance, as the same surgical treatment should be adopted in both cases. On the other hand, simple basilar meningitis often runs a very protracted course. There is often neuritis. The temperature is moderately febrile but frequently high and subject to great fluctuations. Pulse slow and irregular in rhythm until a late stage when it becomes short, quick, and frequent. *Tache cérébrale* distinct. Respirations irregular, shallow, and feeble, and near the end when effusion has taken place into the ventricles the breathing may suddenly cease, the patient become cyanosed, and asphyxia terminate the scene.

In meningitis of the convexity the delirium continues, and with the involvement of the motor centres there are local twitchings or general convulsions; and when there is thrombosis, paresis of the limbs occurs. In basal cases the delirium is also often marked, but in such there are lucid intervals. The most prolonged state of coma-vigil which I have seen occurred in a case of basal meningitis.

When the ventricles are affected the symptoms are somewhat similar to those of the other varieties, such as vomiting,



headache, delirium, and convulsions, but towards the end there are general paretic symptoms with loss of the reflexes, but the cranial nerves are not involved.

In cerebro-spinal fever the symptoms do not differ in any essential particular from those already described. The onset of the disease is abrupt, and the very malignant cases rapidly proceed to suppuration and death, but at least half the cases run a milder course and terminate in recovery. There is early rigidity of the neck, spine, and muscles of the extremities. The head is retracted, and the spine in a more or less marked state of opisthotonos. There is contraction of the flexors and Kernig's symptom is soon developed. The hyperalgesia is pronounced; pain and swelling in the articular joints often occur and may be mistaken for rheumatism. Skin rashes such as erythema, purpuric spots, and herpes labialis are common. In this series there were two cases of cerebro-spinal meningitis, one died and the other after a severe and protracted illness recovered. This latter case was complicated with double otitis interna, which left the patient permanently and absolutely deaf. There was marked vertigo, but he did not complain of it until he was able to be propped up in bed. The erythema and joint affections were prominent features, and even at a late stage there was lymphangitis in one limb, but no suppuration occurred. Hyperalgesia, muscular contractions, and Kernig's sign were all present. There was no optic neuritis. The emaciation was extreme. For a long time there was troublesome incontinence of urine and fæces. It should be noted that in the early stages of meningitis there is often retention of urine.

[Since this address was delivered I have had three cases of cerebro-spinal fever under my care in the Royal Infirmary, and on July 5th I saw another case in consultation with Dr. Main, of Heswall. Dr. Nathan Raw has recently had three cases under his care at the Mill Road Infirmary, so the disease is not quite so rare in this neighbourhood as is generally supposed. Of my cases there have been one male and three females. The male, aged 24, made a good recovery, but the disease was complicated with double otitis interna, and, like the boy previously referred to, he remains absolutely deaf. The two females in the infirmary, aged 24 and 25, died. In both these cases lumbar puncture was performed; in one we found pus and the diplococcus intracellularis; and in the other nothing but clear cerebro-spinal fluid. In the latter case a great many of the joints suppurated, and we found the diplococcus intracellularis in the pus; the cerebro-spinal symptoms subsided, but she died from pyæmia and exhaustion. At the necropsy in this case several of the joints were disorganised and small collections of pus were found in the subarachnoid spaces of the brain and spinal cord, but there was very little injection of the pia, and the inflammatory process seemed to be resolving. In the other necropsy there was pronounced cerebro-spinal inflammation with purulent deposits in the subarachnoid spaces. The case which I saw in consultation with Dr. Main is a remarkable sporadic case occurring in a healthy rural district where there has been no other case and no recent cases of pneumonia. Moreover the woman was not away from home for months.

Mrs. E., aged 41, took suddenly ill on the morning of July 1st, 1899, with severe headache, shivering, and vomiting. When seen by Dr. Main soon afterwards her temperature was

105°. She had not felt quite well for a few days previously, but she ascribed her feelings to menstruation which had occurred for the first time since the birth of her only child five months ago. Probably her surmises were correct, as Dr. Main met her on the road about 36 hours before the commencement of this illness, and she then made no complaint. On July 2nd her temperature dropped to normal, but on the 3rd it was 103°. She then complained of pain in her back and joints, and there was a purpuric rash over her body. Her temperature continued between 101° and 103°, and her pulse ranged between 90 and 130.

July 5th she was much worse, and I saw her with Dr. Main about 1 P.M. She was then complaining of severe pain and tenderness in her spine, right elbow, both knee and ankle joints, which were slightly swollen. Her head was retracted, and any attempt to straighten it or to bring the chin down towards the sternum caused great pain; there was marked opisthotonos and tenderness down the spine especially about the level of the cervical and lumbar enlargements. There was general hyperalgesia; there was much rigidity of the limbs with overaction of the flexors so that the forearms and legs were flexed, and any attempt to straighten them caused pain. Kernig's sign was present. The knee-jerks were absent. There was paralysis of the left third nerve with ptosis, external squint, and diplopia; the left pupil was larger and more fixed than the right; vision good. The *tache cérébrale* was well marked. Temperature 101.6°; pulse 96 full, and tension moderate. Herpes on upper lip, tongue coated with a creamy fur, appetite poor, bowels constipated. Her abdomen flat; she had some difficulty in micturating. Her thoracic and abdominal organs were normal.

She was ordered an ice helmet to be kept constantly applied, cold compresses to the abdomen, a light but liberal nutritious diet, 5 grains of calomel at once, and ⅓ grain of morphine hypodermically every four hours—the morphine to be quickly increased. July 9th. I saw her again this afternoon and found her much better. Her temperature had been on the whole kept well down, but it reached 103.6° on the 7th, and 102.4° on the 8th, and was at my visit 100.4°. Dr. Main rapidly increased the morphine, and for the last two days she has been getting ½ grain every 3 hours. There is now very little pain or tenderness except in the right elbow; tongue clean and moist, appetite better, in fact craving for more food; bowels open. The head less retracted, spine not so rigid, and practically no tenderness; the contractures of the flexors less, and the legs can be almost extended on the flexed thighs; both knee jerks exaggerated and slight right ankle clonus. No swelling in any of the joints. The left ptosis less but still marked external strabismus and diplopia; pupils about equal and not very contracted. We directed the treatment to be continued as before with no diminution of the morphine below 4 grains daily, but if necessary to be increased. She was ordered a fairly liberal diet consisting of Mellin's and Benger's foods made with milk, bread and butter, mutton broth, and white fish, etc.

I went fully prepared to do a lumbar puncture, but I found her so much better that my judgment gained the ascendency over my curiosity, and I left her alone. I cannot say what coccus is the cause of the mischief, but at any rate it is a well-marked case of cerebro-spinal meningitis.

As I was away on my holidays I did not see this patient again till August 7th. She was then in a dying condition, and succumbed the following day. She was delirious, but her attention could be arrested when asked how she felt, or ordered to put out her tongue. There was, and I believe had been for some time, evidence of involvement of the cerebral ventricles by frequent general convulsive twitchings and abolition of the knee-jerks. The superficial reflexes were exaggerated. Dr. Main told me that he had great difficulty in controlling the temperature, and the morphine had not produced any soporific effect, although he had increased it to two-thirds of a grain hypodermically injected every hour and a-half. When I saw her she had been getting over ten grains a day for some time, and was not then under its influence.]

I am convinced that many other organisms besides the diplococcus intracellularis can give rise to cerebro-spinal meningitis. In a child very recently under my care in the Royal Infirmary with marked cerebro-spinal symptoms, retraction of the head, opisthotonos, hyperalgesia, Kernig's sign, general purpuric rash, and swelling of many joints, we did not discover any micro-organisms by a microscopic examination of the clear cerebro-spinal fluid obtained by lumbar puncture, but Dr. Warrington got a culture of staphylococci—probably an accidental contamination. At the *post-mortem* examination there was general injection of the whole cerebro-spinal pia mater, but no tubercles. There was a tuberculous cavity in the left lung about the size of a walnut. The inflammation was evidently due to a tuberculous toxæmia.

Diagnosis.—It might be supposed that a disease with such pronounced symptoms as meningitis would be always easily recognised, but such is not the case, as many of the most prominent cerebral symptoms are common to this and many other acute diseases. In meningitis the classical symptoms, headache and vomiting, have in my opinion led to more failures to recognise this disease than to success in its early diagnosis, because these symptoms are frequently not more marked here than in typhoid and several other specific fevers. The subjective phenomenon, headache, has been so placed in the forefront by textbook authors that the medical attendant almost expects to see it, and so I may say he could if he would only employ his own powers of observation rather than those of the patient, but because the patient is not shouting "Oh! my head," and writhing in agony the onlooker thinks the headache cannot be that of the textbooks. The patient is really too ill to complain, he knows that the noise of his own voice as well as that of his questioner aggravates his suffering. When a child placidly wants your sympathy for a bad headache you may feel confident that it is not due to meningitis. Vomiting is not always an obtrusive symptom; it is explosive, occurs after food and without any nausea. The patient has a poor appetite, takes little or no food, and so there may not be much vomiting. Among the other early signs may be mentioned moderate pyrexia, delirium, stupor, occasionally general convulsions, irregularity of pupils, retraction of the head, retention of urine, etc. One of the earliest and most persistent symptoms, especially in meningitis of the convexity, is general hyperalgesia; so that when you firmly grasp a limb the patient winces. There is intolerance of light and sound. It has been stated by Gowers that lesions of the cranial nerves are among the most definite and conclusive signs of the disease; but if you wait in most cases for the appearance of such signs before beginning a definite line of treatment, you will have a high mortality. This is a disease which no line of treatment will rapidly abort; and if you recognise the nature of the disease at an early stage, you will have plenty of opportunities of confirming your diagnosis. We have previously referred to the various symptoms according to the site of the disease. We can surmise the nature of the disease from the cause. Purulent meningitis is associated with middle ear disease, necrosis of bone, abscess, septicæmia, and frequently cerebro-spinal meningitis. Simple or plastic meningitis may be connected with pneumonia, rheumatism, or some of the specific diseases. Tuberculous meningitis is most common in children under 10 years, but may occur in adults, especially in association with tuberculous disease of the lungs, up to 40 years of age; after this period it is very rare.

Meningitis is frequently mistaken for typhoid fever, but in the early stages at least there should be no difficulty in differentiating the two diseases. In typhoid fever the patient is dull, apathetic, and listless; and although there may be violent headaches, and later on marked delirium, yet the patient is not specially sensitive to external impressions; there is no intolerance of light or sound, no hyperalgesia, he makes no attempt to remove the sordes from his teeth or lips, and the dried secretion in his nose remains unheeded. In meningitis, and especially in meningitis of the convexity, among the early symptoms are marked general hyperalgesia and augmentation in all the reflexes. The patient is irritable and restless, picks at his nose and lips, throws his arms about in an aimless manner, perhaps gets out of bed without knowing exactly why, roams about, returns again to bed. There is hyperalgesia so that he cannot bear to be handled, and generally intolerance of light and sound. The hyperalgesia is often well marked even in an advanced stage of tuberculous meningitis. The little patient may be lying apparently asleep with eyes half open, but the slightest movement or handling makes him scream out with a piercing shriek. The knee, elbow, and wrist jerks are generally pronounced, and there may be well-marked ankle and knee clonus. At a later stage of the disease, if effusion has taken place into the ventricles and beneath the arachnoid, these signs may disappear. The chief characteristic of the deep reflexes is their great variability —absent or present one day and exaggerated the next. In typhoid fever on the other hand the deep reflexes are only exaggerated in severe cases and at a later stage of the disease.

In meningitis the temperature is very variable, frequently reaches a great height and this followed by a rapid fall, and as a rule it is more easily controlled than that of typhoid fever.

We can also examine the urine with Ehrlich's test, and the blood for Widal's reaction, but in the latter test we can rarely expect any positive evidence before the fifth day of typhoid, and the diagnosis should be made before then.

Pneumonia of the apex might be mistaken for meningitis of the convexity, but a careful examination of the lungs, the respiration, and pulse will differentiate the two affections.

Influenza occasionally assumes a cerebral type, but the head symptoms quickly pass off and the real nature of the disease becomes apparent. The same may be said for many of the other specific diseases, such as typhus fever, measles, and scarlet fever.

In bilateral otitis interna there may be headache, delirium, vomiting, convulsions, vertigo, etc., but when the disease is not associated with meningitis the febrile process soon subsides, and there is no great emaciation, etc.

Meningitis has been mistaken for hysteria, but a careful observation of the temperature and other symptoms should readily serve to differentiate the two diseases.

Lumbar Puncture.—Since Quincke drew attention to the diagnostic importance of an examination of the cerebro-spinal fluid, lumbar puncture has been performed in a considerable number of cases, and not infrequently with a positive result. Among the organisms that may be found may be mentioned tubercle bacilli, the diplococcus intracellularis, streptococci, Fraenkel's pneumococcus, and staphylococci. With a positive result the diagnosis is established, but unfortunately in many cases of even purulent meningitis the fluid is perfectly limpid and free from pus and organisms. This is, however, what might be expected as the effusion is usually in the sub-arachnoid spaces, and therefore a negative result is of no value. Some have even claimed considerable therapeutic value for the operation, but the beneficial results seem to have been usually more imaginary than real. The inflammatory effusion for the greater part is in confined spaces, and not floating freely in the cerebro-spinal canal. Where there is an accumulation of inflammatory fluid in the ventricles of the brain, the foramen of Majendie is generally closed, and the fluid cannot escape into the general arachnoid cavity.

Treatment.—The patient should be submitted to as little reflex disturbance as possible. He should be kept in a dark, carpeted, noiseless room so that few nerve impressions should travel to the sensorium by either the eye or ear. The very injurious effect of noise was well exemplified in the fatal case which I published in 1891, when the relapse which ended in his death began on the night when a patient suffering from delirium tremens and carbolic acid poisoning was unfortunately admitted to the same ward. In these respects private practice has a great advantage over the hospital where we cannot always afford such accommodation. Due attention should also be paid to the cutaneous sensibility; when practicable the patient should lie on a water-bed, and be lightly clothed. His head and shoulders should be raised. Great care should be exercised at all times in the application of hot-water bottles to the extremities, as owing to the vasomotor disturbance blistering readily occurs. The room should be well ventilated, and kept at a uniform temperature of about 60°F.

When there is middle ear disease and we have reason to suspect suppurative meningitis, or an abscess in the cerebellum or temporo-sphenoidal lobe, the mastoid cells should be laid open and the disease followed up to the brain, a large trephine opening be made in the skull. If there be evidence of a local abscess anywhere else in the brain surgical treatment should be adopted. Syphilitic meningitis requires specific treatment, and if there be necrosis of bone the surgeon should be called in.

Many cases of meningitis have recovered under very varied treatment, and even in spite of treatment. Iodide of potassium and mercury seem to be the popular remedies even in cases of tuberculous meningitis, but on what grounds I cannot say, certainly they are not employed in tuberculosis in any other part of the body. When the brain is thus affected I fail to see why it should be considered a citadel to be stormed by random shots more likely to kill the patient than the enemy. These drugs should be reserved for syphilitic meningitis. In meningitis I have no faith in the new antipyretic and analgesic drugs; bromide of potassium I have long discarded, while chloral hydrate, sulphonal, paraldehyde, urethane, and all the newer soporifics are by me discredited.

The following methods of treatment are applicable to tuberculous and all the forms of simple meningitis, even including cerebro-spinal fever. There is no disease in which antipyretic treatment requires to be more promptly and efficiently carried out. Of all the means at our disposal for this purpose I prefer a large ice-cap, or a cap formed of Leiter's tubing with a constant stream of cold water flowing through it, applied to the shaven scalp. The application of cold to the head not only lowers the general temperature, but also moderates the circulation within the cranium. This latter statement is perhaps opposed to the researches of Dr. Leonard Hill who denies that there are any vasomotor nerves supplied to the cerebral vessels, and maintains that the cerebral circulation is regulated by the splanchnic area. This latter statement is no doubt in the main true, but it does not necessarily follow that there is no local regulative nervous mechanism of the cerebral circulation. We all know that a large dose of nitrite makes one feel as if the top of the head were coming off. I recollect once in a case of granular kidney an attack of hemiplegia which I ascribed to one-sided spasm of the vessels supplying the motor corex; the hemiplegia was removed by a few doses of nitro-glycerine. I highly appreciate the valuable work of Dr. Leonard Hill, but I am not bound to accept all his conclusions. However, supposing his views are ultimately established we can still explain the beneficial action of the ice-cap by its effects on the general circulation. In many cases this local application of cold is not sufficient to keep the general temperature down to a fairly normal level, and in that event I apply large cold compresses to the abdomen. The application of cold to the splanchnic area raises the general arterial pressure, but this has a beneficial effect on the cerebral circulation by heightening the velocity without necessarily augmenting the transudation from the vessels. The temperature is usually easily controlled in meningitis, but if there should be any hyperpyrexia it can be quickly reduced by swinging the patient in a hammock, and running a small stream of cold or tepid water over the body. The cold to the head should be continued until the general temperature has been for some time subnormal, and if any pyrexia should afterwards recur it should be immediately combated by resort to the ice-cap.

As before stated I have no faith in the new antipyretic drugs. For general use, especially if there be any chest symptoms, I much prefer the free administration of carbonate and acetate of ammonia. If there be any fear of the formation of thrombi in the cerebral sinuses, two to five minims of liquor ammoniæ fort., well diluted with iced barley water, may be given every hour or two. Such cases, however, are generally septic and require surgical treatment.

In the early stage of the disease I have still some faith in the old-fashioned remedy of a fly blister to the nape of the neck, but I very rarely employ it as it rather interferes with the ice-cap, and adds to the discomfort of the patient. In a late stage, when there is evidence of much cerebral effusion with general paresis, and perhaps subnormal temperature it might be worth while to try the effect of liquor epispasticus painted over the whole scalp.

In simple and even in tuberculous meningitis I prescribe, almost as a routine treatment, internal antiseptics, and in such cases I prefer salicylic acid, salol, or benzo-naphthol. A favourite combination with me, especially when the temperature is above the normal, has been equal proportions of Dover's powder and salol; ten grains of each flavoured and suspended in almond mixture every three hours for an adult. The bowels should be kept open throughout the illness, and of all agents for this purpose I prefer calomel, and occasionally enemata. The vomiting is usually of cerebral origin, and any direct treatment to the stomach is generally of little avail; however a sinapism to the epigastrium, and the internal administration of bicarbonate of soda, sal volatile, and creosote frequently does good. The hiccough, which occurs at a late stage, is best relieved by hypodermic injections of morphine, or morphine and atropine. When there is much cardiac excitement with deficient vascular tone, a combination of opium, digitalis, and quinine will be found useful. When there are convulsions the inhalation of chloroform will prove efficacious. In basilar meningitis, when at a late stage there is a tendency to bulbar paralysis, atropine in small doses should be frequently given.

With the use of local or general blood-letting in this disease I have no experience, and in those cases where such a course might be deemed advisable I would rather try the effect of antimony. Depletion might be useful in the early stage if there were violent action of the heart and increased arterial pressure, but such conditions seldom or never occur.

As in other febrile affections, the nasal secretions become dry, inspissated, and irritating; this can be remedied by a spray of 1 or 2 per cent. of menthol in paroline. An antiseptic mouth wash should be used, or it answers equally well for this purpose to paint the teeth and gums with a mixture of equal parts of glycerine and tincture of iodine. If some be swallowed the antiseptic action will be further extended.

To relieve cerebral excitement, sleeplessness, delirium, and general restlessness, I have gradually arrived at the conclusion that there is no drug equal to opium very freely administered. It is not a specific for meningitis or anything else, but to it more than to anything else I ascribe my marked success in the treatment of this disease. It puts the patient in the most favourable position for getting well, and renders his brain less susceptible or more resistant to the storm which is passing over it.

My views on this question have gradually undergone a process of development. At first I used Dover's powder and salicylic acid, opium and calomel; now I almost exclusively use hypodermic injections of morphine, and prescribe the antiseptic remedy separately, or even discard it. It is most difficult to poison a patient suffering from meningitis with opium. I would not advise you to try to poison him for fear you might succeed, but I wish to emphasise the difficulty of doing so, and I am convinced that there is more danger to the patient in using it sparingly rather than freely. In one of my most successful cases the patient, an adult, received each day for over a fortnight 6 grains of morphine hypodermically injected. As the patient began to improve the quantity was gradually reduced; but, with a too rapid diminution at first, there was a relapse, which was cured by a return to a larger dose. In one little patient, who only weighed 28 lbs., and who eventually died from tuberculous meningitis with tuberculous ulceration of the bowels, the quantity of morphine was gradually increased up to 3 grains a day. On one occasion I asked my house-physician, "How is it that this patient is not under the influence of morphine?" To which he replied, "Is there to be no limit?" And I answered that the only limit should be the quantity necessary to keep the patient constantly under the influence of the drug. I prefer a solution of morphine, as being more reliable and more uniform in strength than the hypodermic tablets.

I have often had a little difficulty with house-physicians in indoctrinating them into my views; they have generally been educated by therapeutists who are well up in the coal-tar series, and taught to avoid opium in this disease. I generally commence in children over 7 years of age with ⅙ grain of morphine hypodermically every four hours, and increase the quantity, and perhaps the frequency, as quickly as may be necessary. For children usually about 2 grains a day suffice, but for adults it may be necessary to give 3, 4, 5, or 6 grains daily. For children under 6 or 7 it is perhaps better to administer opium or morphine by the mouth, and to arrive at the requisite dose is a very simple matter. When there is much vomiting it is often an advantage to combine small doses of atropine with the morphine, but if there be much temperature the atropine had better be avoided. At a late stage, when there is evidence of ventricular effusion with a risk of respiratory paralysis, atropine should be given rather freely.

Treatment should be commenced early; when patients are sent into hospital actually dying we should not be expected to perform miracles. I feel confident that if the nervous system were kept absolutely at rest under a free administration of morphine from an early stage of the disease, the mortality would not be so high as it is at present. These views may seem heterodox to many, as the textbooks do not recommend morphine in meningitis, and the few, such as Sir J. Burdon-Sanderson and Professor Osler, who have employed it in cerebro-spinal fever have merely prescribed it to relieve pain.

Diet.—In all these cases there is impaired nutrition, hence the patients should be well fed with light articles of diet, such as peptonised bread and milk, various kinds of children's food, and other articles of farinaceous diet, vegetable juices, grapes, maltine, bread and butter, etc. I always see that the patient gets as great a variety as possible, and as much nutritious food as can be retained in the stomach.